Evolution of transgenerational immunity in invertebrates

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Abstract: Over a decade ago, the discovery of transgenerational immunity in invertebrates shifted existing paradigms on the lack of sophistication of their immune system. Nonetheless, the prevalence of this trait and the ecological factors driving its evolution in invertebrates remain poorly understood. Here, we develop a theoretical host-parasite model and predict that long lifespan and low dispersal should promote the evolution of transgenerational immunity. We also predict that in species that produce both philopatric and dispersing individuals, it may pay to have a plastic allocation strategy with a higher transgenerational immunity investment in philopatric offspring because they are more likely to encounter locally adapted pathogens. We review all experimental studies published to date, comprising 21 invertebrate species in 9 different orders, and we show that, as expected, longevity and dispersal correlate with the transfer immunity to offspring. The validity of our prediction regarding the plasticity of investment in transgenerational immunity remains to be tested in invertebrates but also in vertebrate species. We discuss the implications of our work for the study of the evolution of immunity and we suggest further avenues of research to expand our knowledge of the impact of transgenerational immune protection in host-parasite interactions.

Key words: transgenerational immune effect, local adaptation, dispersal, longevity, Drosophila, eusocial insects.

1. Introduction

The immunity of invertebrates was, for a long time, widely assumed to lack the most sophisticated component of the vertebrate immune system: its ability to mount an acquired response where memory effectors produced during an infection protect the individual (within-generational protection) or its offspring (trans-generational protection) against subsequent infections. Yet, recent research has shown that invertebrates have spectacularly plastic immune effectors that can generate true novelty and functional immune responses following exposure to pathogens [1,2]. Experimental evidence of the existence of within-generational immune priming in invertebrates has grown considerably in the last decade [3,4]. It has been documented in a range of invertebrate species including *Decapoda* [5], *Branchiopoda* [6], *Lepidoptera* [7], *Coleoptera* [8], *Diptera* [9] and *Hymenoptera* [10]. Interestingly, in some cases immune priming has been shown to persist not only throughout the lifespan of the animal [11,12] but also across generations [13–15]. Transgenerational immunity has been thus far reported in a dozen invertebrate species [13,14,16–24]. Although the mechanisms underlying this transgenerational immune protection remain unclear, current work suggests that this form of parental care may be induced by the transfer of pathogen-derived antimicrobial peptides or mRNA-encoding immune effectors [20,25,26].

Transgenerational immune protection potentially confers a large fitness advantage to offspring [13]. This form of parental protection, however, does not seem to be widespread amongst invertebrates. Indeed, several studies have failed to detect any transgenerational transfer of immunity [27–30], and others have even found a negative impact of maternal infection on offspring resistance to pathogen infections [31]. This raises the question of what are the conditions that favour the evolution of transgenerational immunity in invertebrates.

In this study, we investigate whether the presence or absence of transgenerational immune protection in invertebrates is explained by factors related to the biology and the ecology of the species. For this purpose we first modify the theoretical approach developed by Garnier *et al* (2012) [32] for a single host population, by considering two invertebrate host populations connected by migration. Each host population is exposed to a different pathogen and migrating hosts have varying degrees of cross immunity to the resident parasite. We study the impact of host dispersal, host lifespan, immunity costs, force of infection and parasite virulence on the evolution of transgenerational immunity. We then confront the predictions issued from these models to currently available data. For this purpose, we review all experiments published to date on transgenerational immunity or transgenerational protection in invertebrates, focusing in particular on two traits for which information is readily available at the species level: average dispersal and lifespan. To our knowledge, our study is the first attempt to confront theoretical predictions with empirical patterns of transgenerational immunity in invertebrate species.

2. Materials and Methods

Theoretical analysis

The evolution of maternal transfer of immunity has been studied elsewhere in a single host population [32,33]. Here we expand these previous models and we study the evolution of maternal transfer of immunity in invertebrates in a habitat with two populations connected by migration. Each population is assumed to be exposed to a different pathogen and the pathogen is not allowed to migrate between populations, which maximizes the heterogeneity of the environment. In population *i* (where *i* = 1 or 2) susceptible individuals, S_i, are exposed to a constant rate of infection h_i which yields

infected individuals, I_i. All individuals die naturally, with rate μ, and infected individuals suffer additional parasite related mortality (i.e. virulence), with rate α. All individuals can produce offspring that can move to a different patch, with probability of dispersal η. We assume that infected individuals can transmit transient immunity to their offspring against the parasite they are infected with. We assume that the investment in immunity transfer may be modulated by the dispersal phenotype of the offspring. The probability of immunity transfer is θ_P and θ_D for philopatric and dispersed individuals, respectively. We also consider a scenario where immunity transfer, θ, is not allowed to vary between philopatric and dispersed offspring. The ability to transfer immunity is further assumed to be associated with a fecundity cost c_θ. We keep track of the origin of the maternally protected individuals using the notation M_{ij} for the density of maternally protected individuals produced in population *i* and currently in population *j* (where *i* and *j* = 1 or 2). Hence M_{ij} is immune to parasites from population *i* but only partially immune to pathogens from population *j*. The amount of cross immunity is governed by the parameter χ and the force of infection on M_{ij} is $(1-\chi)\, h_j$, with $0 \leq \chi \leq 1$. Maternal protection is assumed to be transitory and it wanes at rate δ_M in all populations. We use this model to study the effect of various ecological scenarios on the evolutionary stable investment in transgenerational immunity (see the Supplementary Information for mathematical details).

Empirical data: transgenerational effect scores

To test our theoretical predictions, we carried out an extensive literature review that included all the papers on transgenerational immune priming or transgenerational offspring protection in invertebrates published to date (summarised in table Table S1). This consisted of 35 published articles comprising a total of 21 invertebrate species. We identified two different protocols for measuring transgenerational immune priming. Some studies,

117 investigate the impact of either parental infection or immune stimulation on offspring
118 immunity (we henceforth call this **TEI**, for **Transgenerational Effect on Immunity**). These
119 studies quantify and compare immune priming by measuring different immune parameters
120 (melanisation, phenoloxidase [PO] production, antibacterial peptide production, haemocyte
121 number and immune transcripts) in offspring issued from immune stimulated and naïve
122 parents. For simplicity, we scored these studies as either 1 (offspring of infected parents
123 have an increased production of least one of the immune effectors) or 0 (offspring of
124 infected parents have similar or lower production of a given immune effector). When
125 different studies have been carried out on the same species, the overall TEI score for the
126 species was obtained by averaging across studies. Second, we identified another set of
127 studies where both parents and offspring are exposed to live pathogens. These studies
128 record immune priming by quantifying the outcome of an infection (parasite prevalence,
129 parasite intensity or survival) in offspring issued from infected and uninfected parents (**TER**
130 for **Transgenerational Effect on Resistance**). As above, these studies were scored as either 1
131 (offspring from infected parents have lower parasite prevalence, lower intensity or higher
132 survival than offspring from naïve parents) or 0 (when the opposite, or when no effect of
133 parental infection is observed), and the average score for the species was obtained by
134 averaging across studies. Finally, for each species we obtained an overall measure of
135 investment in offspring protection (**OTP** for **Overall Transgenerational Protection**) which
136 was scored as 1 when either TEI or TER (or both) were 1, and 0 otherwise.

137 For each species, we focused on two ecological parameters for which there is
138 available information in the literature: lifespan and dispersal. We define dispersal, as the
139 average distance travelled by adults, in most cases estimated using mark and recapture
140 methods in the field, and lifespan as the average longevity of a species estimated under
141 standard laboratory conditions. Although both parameters are known to vary widely

according to environmental and experimental conditions (eg, nutrition, temperature), these studies provide ballpark estimates of the dispersal (0 - 6600 metres) and longevity (24 - 700 days) ranges across species. In three species, no data about dispersal was available in the literature and therefore this analysis was performed on a subset of 18 species.

Statistical analysis

All statistical analyses were performed using the software R (v.3.1.0, http://www.cran.r-project.org/). In order to compare TEI, TER, and OTP we first carried out a Fisher's exact test using longevity and dispersal as categorical variables. Species were classified as having a short (< 60 days) or long (≥ 60 days) lifespan, and those with a short (< 500 m) and long (≥ 500m) dispersal range. We controlled the robustness of our analyses by using several different cut-off points for defining short and long lifespan and dispersal range (9 points for longevity and 8 for dispersal, Figure S1). Fisher's exact test, however, obviates the fact that species are phylogenetically related and are therefore not statistically independent units. In order to account for this phylogenetic signal we performed a second analysis using a linear regression for binary phylogenetic data (binary PGLMM, packages 'ape', [34]). Phylogenetic information (Figure S2) for the 21 species was obtained from the Interactive Tree of Life (http://itol.embl.de). The branch lengths were obtained from the Timescale of Life (http://timetree.org) and from Niklas Wahlberg (personal communication) for Lepidoptera species.

3. Results

Theory

We explored the effect of the different parameters of the model on the evolution of the maternal transfer of immunity. As expected, we show that increasing the force of

infection h or decreasing the cost c_θ associated with the transfer of immunity always selects for higher values of θ. As pointed out by Garnier et al. (2012) [32] pathogen virulence has a non-monotonic effect on the evolution of θ. Both avirulent and very virulent pathogens select for low levels of maternal transfer of immunity. Indeed, when virulence becomes very high it is not worth investing in a resistance mechanism that will never be expressed as infected individuals have very little opportunity to reproduce before they die from the infection. High levels of investment in θ are only selected when pathogens induce an intermediate reduction in longevity. We also retrieve the effect of longevity discussed in Garnier et al. (2012) [32]. Short lived species do not invest in transgenerational immunity because the survival benefit associated with immunity is cancelled out by the intrinsic mortality rate, μ (**Figure 1A**).

In addition our model allowed us to explore the effect of dispersal and cross immunity on the evolutionary outcome. When dispersal is high and cross immunity is low maternal investment is unlikely to protect the offspring because they are likely to be exposed to a different pathogen. Consequently, higher investment in maternal transfer is only expected to evolve in philopatric species or in species with high levels of cross immunity (**Figure 1B**). In the case where mothers have the ability to produce both philopatric and dispersing offspring and cross immunity is imperfect, maternal investment is predicted to be higher in the philopatric progeny (i.e. $\theta_P > \theta_D$, **Figure 2**). Indeed, such plastic investment in transgenerational immunity is adaptive because philopatric offspring are more likely to be exposed to the same pathogens.

Empirical data

We focused our attention only on two key life history traits of the host for which sufficient information is available in the literature: lifespan and dispersal. We investigated

190 the impact of these two parameters in each of the transgenerational immunity scores
191 identified above.

192 As expected, long-lived species and species with short dispersal ranges have
193 significantly higher TER scores (respectively Fisher exact Test, p = 0.039, **Figure 3A,** p = 0.017,
194 **Figure 3B**). Neither longevity (Fisher Exact Test, longevity: p = 0.318) nor dispersal range (p =
195 0.444) have a significant effect on the TEI scores (**Figure 3C**, **3D**). Interestingly, however,
196 both dispersal and lifespan have a significant impact on the overall parental investment in
197 offspring protection as quantified by the OTP score (**Figure 3E, 3F**). Species with long lifespan
198 and short dispersal ranges have significantly higher OTP scores than their short-lived and
199 highly dispersing counterparts (Fisher Exact Test, lifespan: p=0.002, dispersal: p=0.047). The
200 effect of lifespan on the OTP score is largely robust with respect to the cut-off point between
201 long and short lived species (**Figure S1A**). Dispersal, however, is highly sensitive to the cut-
202 off point chosen, and significance is lost in all but the 500 cut-off point (**Figure S1B**).

203 To verify whether results hold when correcting for phylogenetic correlations, the
204 analyses were repeated using linear regression for binary phylogenetic data. In accordance
205 with the results of the Fisher Exact Test lifespan has a significant effect on the OTP score
206 (cut-off point: 60 days, Zscore= 2.031, p = 0.042); dispersal, however, loses its significance at
207 the 500 m cut-off point (Zscore= -0.617, p = 0.537).

208 4. **Discussion**

209 Previous work has shown how investment in immunity, and in classic (within-
210 generational) immune memory in particular, should be maximized in species with high or
211 intermediate lifespan [4,35–38]. Simply put, short lived hosts are unlikely to encounter the
212 same pathogen twice and should therefore not invest in memory. Recently Garnier et al.
213 (2012) [32] and Metcalf & Jones (2015) [33] showed that these predictions could be also

extended to the evolution of the maternal transfer of immunity in a single host population. Here, we consider a scenario with two host populations connected by migration. In addition, we assume that infected hosts cannot recover from the infection (as is the case in most invertebrates) but may be able to transfer some immunity to its offspring. Our results agree with previous studies in showing that the marginal gain in fitness obtained from transgenerational immunity is higher in long-lived species. Our prediction is supported by empirical data confirming the existence of an association between transgenerational immunity and longevity in invertebrates: immune-challenged long-lived species have a higher probability of actively protecting their offspring against a subsequent infection than their short-lived counterparts.

The amount of host dispersal is expected to affect the evolution of host-parasite interactions and in particular to shape patterns of parasite local adaptation [39–42]. Since parasites are often found to be adapted to their sympatric hosts [40], host migration may reduce the cost of parasitism and could affect the evolution of immunity [43–45]. For instance, Kurtz et al. [46] showed that after being placed into a new environment the grasshopper (*Chorthippus biguttulus*) reduces the expression of a non-specific immune trait (*i.e.* phagocytosis activity), possibly due to a lower exposure to locally adapted parasites. In the present study we focus on the evolution of immune transfer under the assumption that parasites are locally adapted, and we show that philopatry can promote the evolution of transgenerational immunity because it increases the predictability of the offspring environment. In other words, maternal transfer of protection should be favoured when mothers and offspring share the same environment and are thus likely to be exposed to similar parasites. This prediction, however, could not be satisfactorily confirmed using currently available data. Dispersal is only a marginally significant predictor of maternal

transfer of immunity at one of the cut-off points (500 m) and the significance is lost when the phylogeny is taken into account in the analysis.

Broadly speaking, our ability to test our theoretical predictions concerning dispersal and longevity was limited not only by the difficulties inherent to quantifying these parameters in wild invertebrates but also by the limited number and phylogenetic breadth of taxa in which transgenerational immune priming has been quantified to date. Transgenerational immunity has thus far been described in a mere dozen invertebrate species, the large majority of which are either aquatic, eusocial or stored-product species (Table S1). This problem is, we suspect, compounded by a publication bias that favours the publication of significant results over non-significant ones. Expanding the range of transgenerational immune protection studies to a large panel of invertebrate taxa with a wide range of life history traits is an essential first step to understanding the ecological conditions under which this trait evolves. Terrestrial isopod species are good candidates due to their limited dispersal potential [47] and extended lifespans, which can range between one to more than five years, depending on the species [48–50]. The confounding effect of phylogeny could be bypassed by working with taxa displaying a range of different life history traits, such as the bee super-family of *Apoidea* which contains both eusocial and solitary bees. Finally, experimental evolution mimicking different ecological scenarios (eg high/low dispersal) could provide a powerful tool to test some of these predictions using laboratory-friendly species (e.g. *Drosophila, Artemia*).

Our theoretical model also generates testable predictions on the evolution of a plastic transfer of immunity in species that can produce both dispersing and non-dispersing morphs. Under the assumption that parasites are locally adapted and that immunity is specific (i.e. that there is low cross immunity) mothers are expected to invest more in the

immune protection of the philopatric, non-dispersing morph, than on the dispersing one. This prediction could be tested in insects producing both apterous and winged (alate) forms, such as aphids [51–53], ants [54, 55] and termites [56], or in species that exhibit a sex-biased dispersal, such as gypsy moths [57] and midges [57]. In each of these cases the philopatric morph or sex is expected to accrue greater benefits from a higher maternal investment in immunity than the dispersing one. Incidentally, this prediction could be validated in vertebrates, such as certain bird and mammal species that exhibit drastic differences in sex-biased dispersal [58]. Finally, our predictions may have implications for when dispersal happens across time rather than space, as is the case in species that produce dormant stages. Dormancy may favour the evolution of conditional investment in immunity: dormant offspring are often expected to be exposed to maladapted pathogens [59, 60] and may require lower investment in immunity than their non-dormant counterparts.

Our review of the experimental literature revealed broad methodological differences between the studies which raise both conceptual and terminological issues regarding what constitutes transgenerational immunity. Two different protocols are used to test for transgenerational immunity and they do not necessarily convey the same information. About half of the studies quantify and compare immune priming by measuring a handful of immune parameters in offspring from immune stimulated and naïve parents (TEI), but do not necessarily verify whether the increased immune effectors result in increased parasite protection. The use of a few (typically one or two) immune assays as a proxy for parasite resistance has come under increased scrutiny, as evidence accumulates that they are not necessarily correlated with each other [61]. In other words, an elevated TEI, does not necessarily imply either that the mother pays any costs for the transfer (immune effectors could diffuse passively into eggs within the ovaries), or indeed that the offspring are better

protected as a result (if for example immune components are not transmitted in sufficient numbers). Conversely, the other half of the studies, quantify the outcome of an infection (parasite prevalence, parasite intensity or survival) in offspring issued from infected and uninfected parents (**TER)** but without delving into whether the underlying mechanisms are immunological or not (for example through the differential provisioning of offspring with nutritional resources). Our analyses showed that while the results obtained from TER studies are largely consistent with our theoretical predictions, the signal is much less clear for TEI studies. We believe that an integrative view of the transgenerational immune memory requires both approaches [17,18,21,62,63].

In conclusion, there is a growing interest on the biology and ecology of transgenerational immune priming in invertebrates [64], not least due to the key role some of them play as pollinators, vectors of diseases, and agricultural and stored product pests. Transgenerational immune priming is predicted to have not only a strong effect on disease prevalence [65,66] but also on the age structure [65] and population dynamics of invertebrates [66]. Our theoretical model shows that, beyond the effect of host lifespan and host dispersal, several other life history parameters play a key role in the evolution of transgenerational immunity. Future work needs to expand on currently available data in order to get a wider picture of the transgenerational immune protection and on its impact on the evolutionary ecology of the host-pathogen interactions.

Data accessibility

The complete data can be found in the supplementary information

Competing interests

We declare we do not have any competing interest.

Authors' contributions.

R.G. and S.G. developed and analysed the theoretical model, R.P. reviewed and analysed empirical data, R. P. S.G. A. R. wrote the paper.

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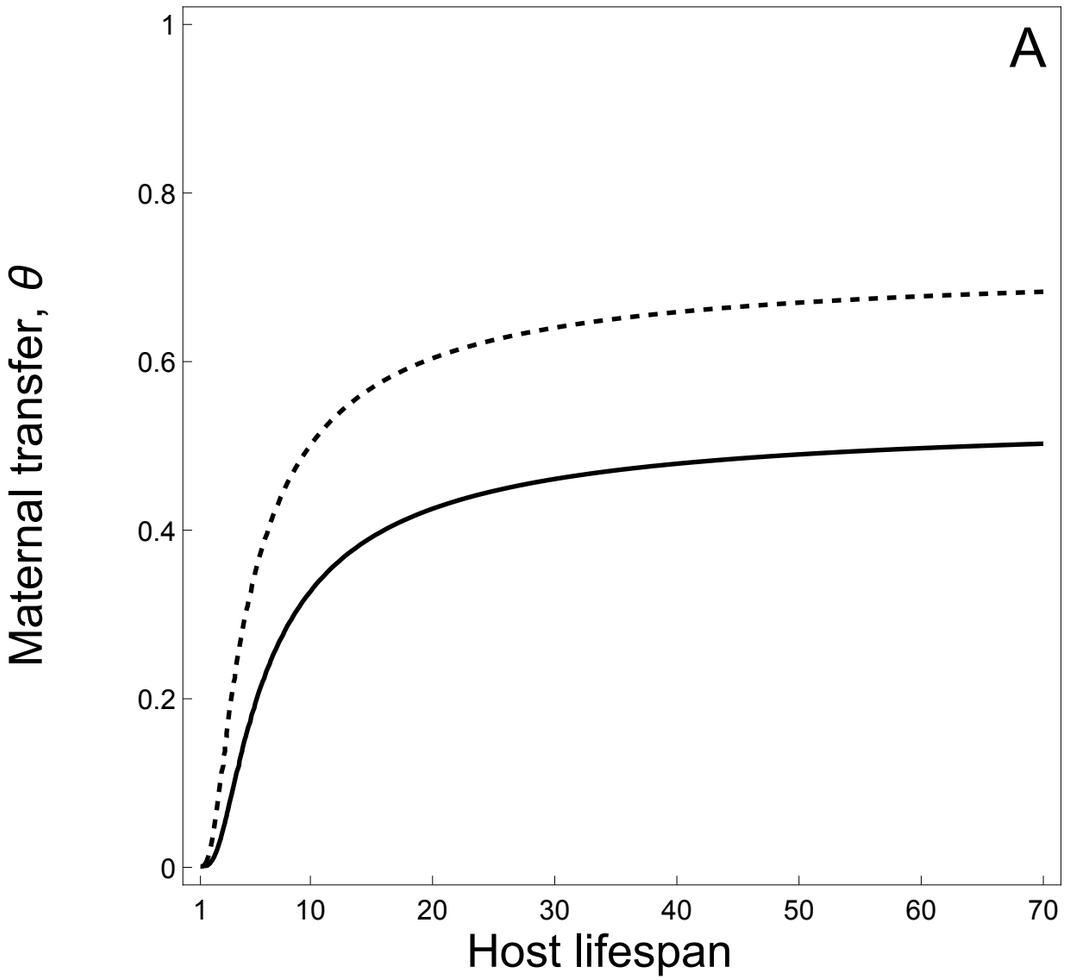
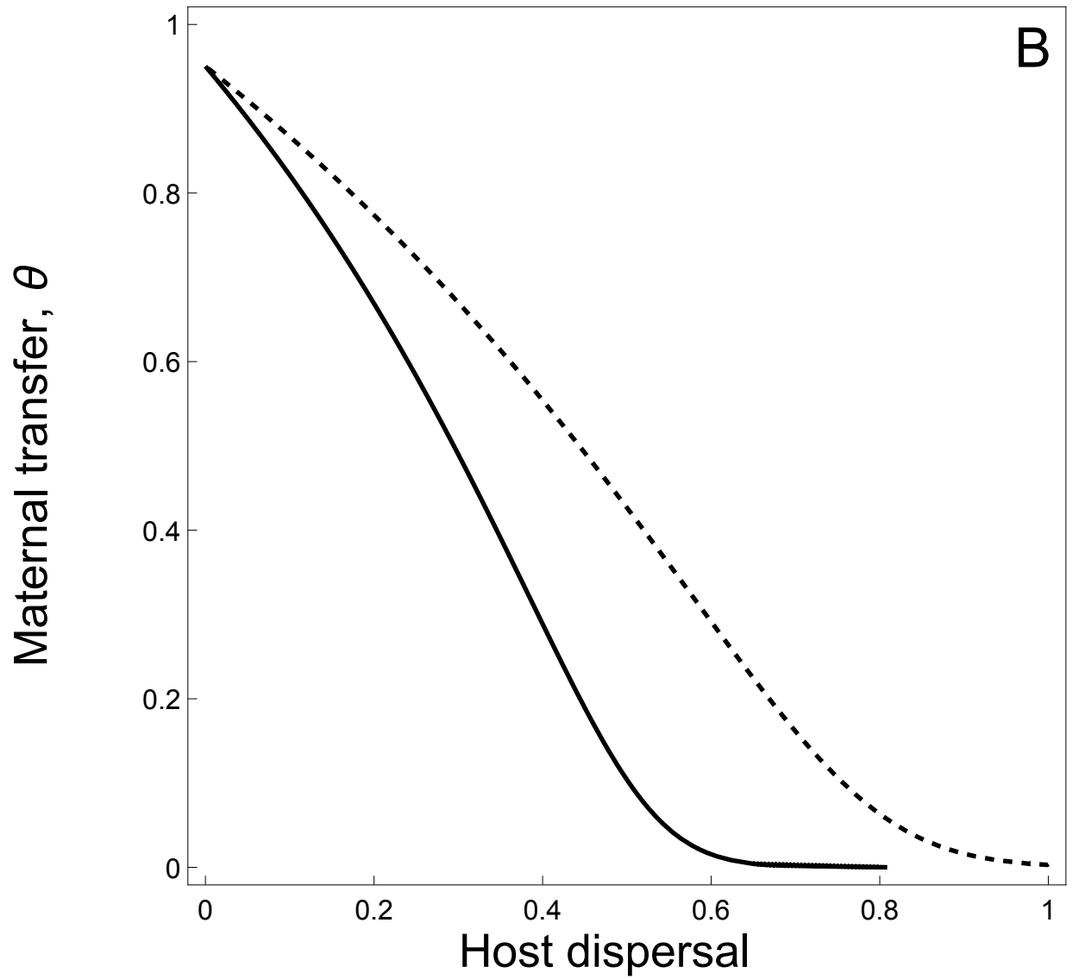
Figure legends

Figure 1: Evolutionary stable investment in maternal transfer of immunity θ (when $\theta_P = \theta_D$) with or without cross-immunity: $\chi = 0.5$ (dashed line) and $\chi = 0$ (full line) against (A) the longevity of the host, (B) the dispersal of the host. Default parameter values (see supplementary material for more details on the model) : $r_0 = 1.5$, $c_\theta = 0.1$, k=1.1, $\eta = 0.3$, $K = 20$, $h = 1.1$, $\alpha = 3$, $\delta_M = 1$, $\mu = 0.02$.

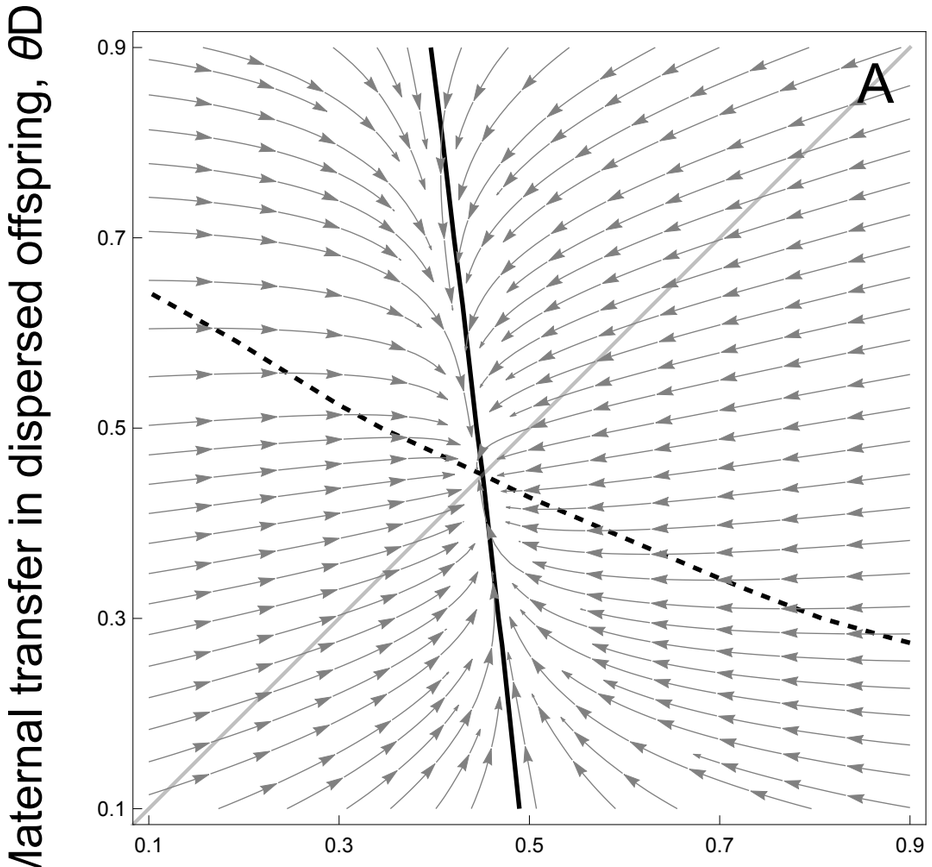
Figure 2: Evolution of maternal transfer of immunity towards philopatric θ_P or dispersed offspring θ_D (A) with perfect cross-immunity $\chi = 1$ or (B) with imperfect cross immunity. The full line is the evolutionary stable value of θ_P against θ_D and the dashed line is the evolutionary stable value of θ_D against θ_P. The intersection between these two lines indicates the coevolutionary stable strategy of θ_D and θ_P. The arrows indicate the direction of evolution on both these traits. Default parameter values (see supplementary material for more details on the model) : $r_0 = 2$, $c_\theta = 0.2$, k=1.5, $\eta = 0.25$, $K = 20$, $h = 1$, $\alpha = 3$, $\delta_M = 1$, $\mu = 0.02$.

Figure 3: Scores for Transgenerational Effect on Resistance (TER, white bars), Transgenerational effect on Immunity (TEI, grey bars) and Overall Transgenerational Protection (OTP, black bars) according to species longevity (A) and dispersal (B). Statistical analyses were performed separately for each group (TER, TEI and OTP). Levels not connected by same letter are significantly different. Error bars represent ±SE.

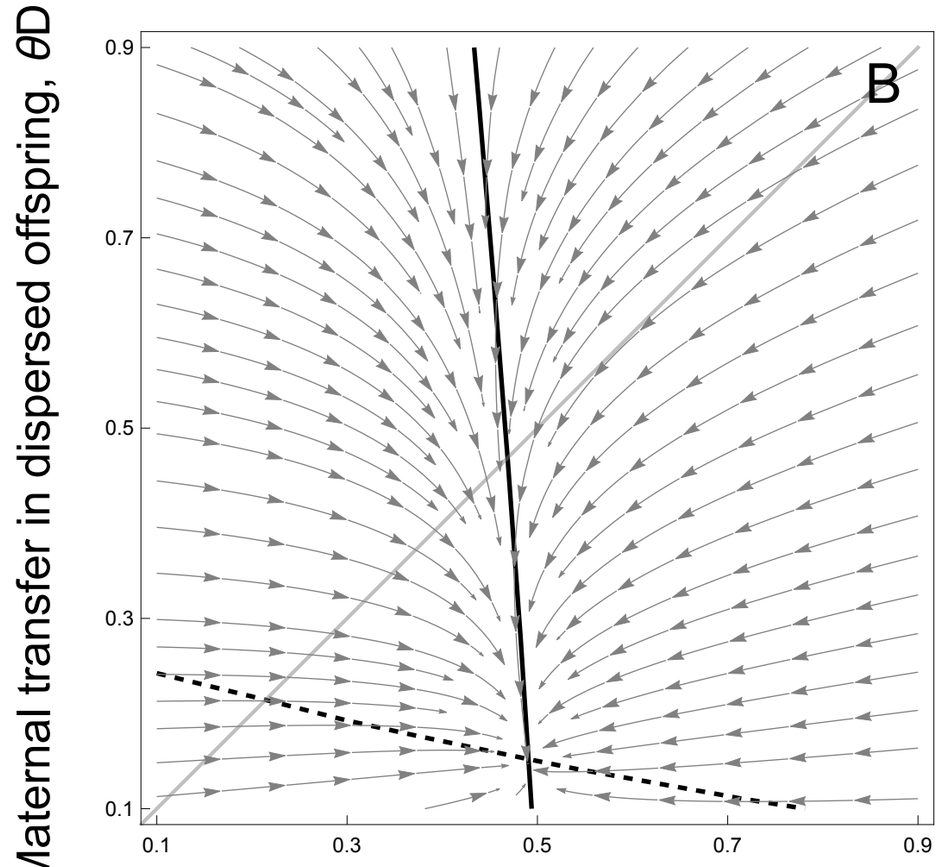

A
Maternal transfer, θ
1
0.8
0.6
0.4
0.2
0
1
10
20
30
40
50
60
70
Host lifespan

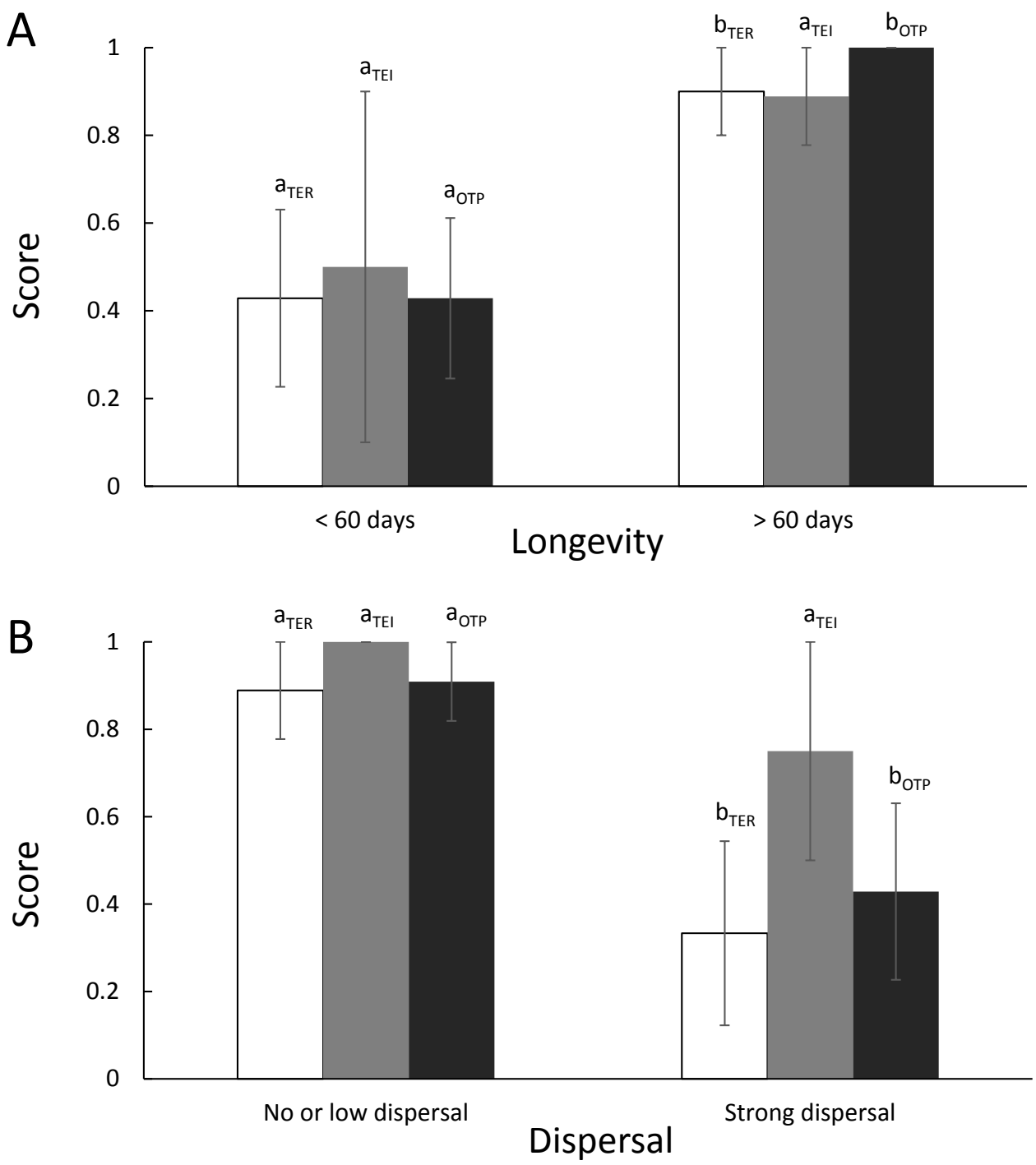

B
Maternal transfer, θ
1
0.8
0.6
0.4
0.2
0
0
0.2
0.4
0.6
0.8
1
Host dispersal

Cross immunity χ=1



Cross immunity χ=0.5




A
Score
1
0.8
0.6
0.4
0.2
0
a_{TER}
a_{TEI}
a_{OTP}
b_{TER}
a_{TEI}
b_{OTP}
< 60 days
> 60 days
Longevity
B
Score
1
0.8
0.6
0.4
0.2
0
a_{TER}
a_{TEI}
a_{OTP}
a_{TEI}
b_{TER}
b_{OTP}
No or low dispersal
Strong dispersal
Dispersal

Evolution of transgenerational immunity in invertebrates

Supplementary materials

Theoretical analysis

Following Garnier et al. (2012) we focus on the ability of a mutant host to invade a resident host population at equilibrium. The pathogen is assumed to impose a constant force of infection h_i in host population i. The transfer of immunity is allowed to differ between philopatric offspring (θ_P^m for the mutant and θ_P for the resident) and dispersed offspring (θ_D^m for the mutant and θ_D for the resident). But we also consider a case where the host is not allowed to adopt a conditional strategy. In this case there is a single strategy for the mutant (i.e., $\theta_P^m = \theta_D^m = \theta^m$) and a single strategy for the resident (i.e., $\theta_P = \theta_D = \theta$). The mutant may be present in different populations and in different host states. The densities of the different types of hosts are given in the vector $H^m = (M_{11}^m, M_{21}^m, S_1^m, I_1^m, M_{22}^m, M_{12}^m, S_2^m, I_2^m)^T$. The dynamics of the mutant can be fully described by the matrix $\boldsymbol{F}^m$ which accounts how many mutants are created in the 6 different host types and the matrix $\boldsymbol{V}^m$ which refers to transition between these types:

$$\boldsymbol{F}^m = \begin{pmatrix} 0 & 0 & 0 & \theta_P^m(1-\eta)\lambda_1^m & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_D^m\,\eta\,\lambda_1^m \\ (1-\eta)\lambda_1^m & (1-\eta)\lambda_1^m & (1-\eta)\lambda_1^m & (1-\theta_P^m)(1-\eta)\lambda_1^m & \eta\lambda_2^m & \eta\lambda_2^m & \eta\lambda_2^m & \eta(1-\theta_D^m)\lambda_2^m \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_P^m(1-\eta)\lambda_2^m \\ 0 & 0 & 0 & \theta_D^m\,\eta\,\lambda_2^m & 0 & 0 & 0 & 0 \\ \eta\lambda_1^m & \eta\lambda_1^m & \eta\lambda_1^m & \eta(1-\theta_D^m)\lambda_1^m & (1-\eta)\lambda_2^m & (1-\eta)\lambda_2^m & (1-\eta)\lambda_2^m & (1-\theta_P^m)(1-\eta)\lambda_2^m \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{(S1)}$$

$$\boldsymbol{V}^m = \begin{pmatrix} \delta_M+\mu & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta_M+\mu+(1-\chi)h_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\delta_M & -\delta_M & \mu+h_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(1-\chi)h_1 & -h_1 & \mu+\alpha & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \delta_M+\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \delta_M+\mu+(1-\chi)h_2 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\delta_M & -\delta_M & \mu+h_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -(1-\chi)h_2 & -h_2 & \mu+\alpha \end{pmatrix} \quad \text{(S2)}$$

where, $\lambda_i^m = r_0\Big(1 - c_\theta((1-\eta)(\theta_P^m)^k + \eta(\theta_D^m)^k)\Big)(1 - N_i/K_i)$ is the fecundity of mutant hosts. As in Garnier et al. (2012) the maximal fecundity r_0 is reduced by the cost associated with the mutant strategy θ_P^m and θ_D^m (where c_θ affects the magnitude of the cost and k the

shape of the relationship between the investment in maternal transfer of immunity and the cost) and by the density dependence in each host population *i*.

We can further simplify this model under the additional assumption that the two populations are symmetric: $h_1 = h_2 = h,\ K_1 = K_2 = K,\ N_1 = N_2 = N,\ \lambda_1^m = \lambda_2^m = \lambda^m$. Because of the symmetry we can focus on a single population *i* and on the vector $H_i^m = \left(M_{ii}^m, M_{ji}^m, S_i^m, I_i^m\right)^T$. The dynamics of the mutant can be derived fom the matrices $\boldsymbol{F}_i^m$ and $\boldsymbol{V}_i^m$:

$$\boldsymbol{F}_i^m = \begin{pmatrix} 0 & 0 & 0 & \theta_P^m(1-\eta)\lambda^m \\ 0 & 0 & 0 & \theta_D^m\,\eta\,\lambda^m \\ \lambda^m + \delta_M & \lambda^m + \delta_M & \lambda^m & \left((1-\eta)(1-\theta_P^m) + \eta(1-\theta_D^m)\right)\lambda^m \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{S3}$$

$$\boldsymbol{V}_i^m = \begin{pmatrix} \delta_M + \mu & 0 & 0 & 0 \\ 0 & \delta_M + \mu + (1-\chi)h & 0 & 0 \\ 0 & 0 & \mu + h & 0 \\ 0 & -(1-\chi)h & -h & \mu + \alpha \end{pmatrix} \tag{S4}$$

The per-generation growth rate of the mutant is given by the dominant eigenvalue of the matrix $\boldsymbol{B}_i^m = \boldsymbol{F}_i^m.\boldsymbol{V}_i^{m\,-1}$ which is:

$$\boldsymbol{B}_i^m = \begin{pmatrix} 0 & \tau_{M_{ji}\to M_{ii}}^m & \tau_{S_i\to M_{ii}}^m & \tau_{I_i\to M_{ii}}^m \\ 0 & \tau_{M_{ji}\to M_{ji}}^m & \tau_{S_i\to M_{ji}}^m & \tau_{I_i\to M_{ji}}^m \\ \tau_{M_{ii}\to S_i}^m & \tau_{M_{ji}\to S_i}^m & \tau_{S_i\to S_i}^m & \tau_{I_i\to S_i}^m \\ 0 & 0 & 0 & 0 \end{pmatrix} \tag{S5}$$

where $\tau_{X\to Y}^m$ is the per-generation transition between host type X and Y:

$$\tau_{M_{ji}\to M_{ii}}^m = \frac{(1-\eta)h\lambda^m(1-\chi)\theta_P^m}{(\alpha+\mu)(\delta_M + h(1-\chi) + \mu)}$$

$$\tau_{S_i\to M_{ii}}^m = \frac{(1-\eta)h\lambda^m\theta_P^m}{(h+\mu)(\alpha+\mu)}$$

$$\tau_{I_i\to M_{ii}}^m = \frac{(1-\eta)\lambda^m\theta_P^m}{\alpha+\mu}$$

$$\tau_{M_{ji}\to M_{ji}}^m = \frac{\eta\theta_D^m}{(1-\eta)\theta_P^m}\tau_{M_{ji}\to M_{ii}}^m$$

$$\tau_{S_i\to M_{ji}}^m = \frac{\eta\theta_D^m}{(1-\eta)\theta_P^m}\tau_{S_i\to M_{ii}}^m$$

$$\tau^m_{I_i \to M_{ji}} = \frac{\eta \theta_D^m}{(1-\eta)\theta_P^m} \tau^m_{I_i \to M_{ii}}$$

$$\tau^m_{M_{ii} \to S_i} = \frac{\lambda^m + \delta_M}{\delta_M + \mu}$$

$$\tau^m_{M_{ji} \to S_i} = \frac{\delta_M(\alpha+\mu) + \lambda^m(\alpha + h(1-(1-\eta)\theta_P^m - \eta\theta_D^m)(1-\chi) + \mu)}{(\alpha+\mu)(\delta_M + h(1-\chi) + \mu)}$$

$$\tau^m_{S_i \to S_i} = \frac{\lambda^m(h(1-(1-\eta)\theta_P^m - \eta\theta_D^m) + \alpha + \mu)}{(h+\mu)(\alpha+\mu)}$$

$$\tau^m_{I_i \to S_i} = \frac{(1-(1-\eta)\theta_P^m - \eta\theta_D^m)\lambda^m}{\alpha+\mu}$$

The dominant eigenvalue of this matrix is of the form:

$$R_m = \frac{A + \sqrt{B}}{2} \tag{S6}$$

with :

$$A = \tau^m_{S_i \to S_i} + \frac{\eta \theta_D^m}{(1-\eta)\theta_P^m} \tau^m_{M_{ji} \to M_{ii}}$$

$$B = \left(\tau^m_{S_i \to S_i} - \frac{\eta \theta_D^m}{(1-\eta)\theta_P^m} \tau^m_{M_{ji} \to M_{ii}} \right)^2 + 4\tau^m_{S_i \to M_{ii}} \left(\tau^m_{M_{ii} \to S_i} + \frac{\eta \theta_D^m}{(1-\eta)\theta_P^m} \tau^m_{M_{ji} \to S_i} \right)$$

As expected, in the absence of dispersal between population (i.e. $\eta = 0$), we recover the dominant eigenvalue (A9) derived in Garnier et al. (2012).

Table S1 : summary transgenerational protection

	Refs	Immune stimulation (F0)	Immune stimulation (F1)	Increased resistance?	TER	Effects on immunity (F1)	TEI	Age	Active dispersal	OTP
Molusca										
Chlamys farreri	[1]	HK Bacteria	Bacteria	YES (survival)	1	YES (ab activity, Immune-related proteins)	1	> 60 days [36]	ni	1
Crustacea					-		-			
Penaeus sp.	[2]	Glucane	Virus	YES (survival)	1	*ni*	-	> 60 days [37]	ni	1
Brachiopoda							-			
Daphnia magna	[3]	Bacteria	same as F0	YES (prevalence)	1	*ni*	-	≤ 60 days [38]	No or low dispersal [56]	1
Artemia	[4]	Bacteria	same as F0	YES (survival)	1	*ni*	-	> 60 days [39]	No or low dispersal [57]	1
Artemia	[5]	Bacteria	same as F0	YES (survival)	1	YES (Immune transcripts)	1	> 60 days [39]	No or low dispersal [57]	1
Hymenoptera										
B. terrestris	[6]	LPS	none	*ni*	-	YES (PO activity)	1	> 60 days [40]	No or low dispersal [58]	1
B. terrestris	[7]	HK bacteria	LPS	*ni*	-	YES (ab activity)	1	> 60 days [40]	No or low dispersal [58]	1
B. terrestris	[8]	HK bacteria	same as F0	*ni*	-	YES (ab activity)	1	> 60 days [40]	No or low dispersal [58]	1
B. terrestris	[9]	HK bacteria	HK bacteria/Tripanosoma	NO (burden*)	0	YES (ab activity)	1	> 60 days [40]	No or low dispersal [58]	1
B. terrestris	[10]	HK bacteria	same as F0	*ni*	-	YES (Immune transcripts)	1	> 60 days [40]	No or low dispersal [58]	1
A. mellifera	[11]	Bacteria	none / same as F0	YES (survival)	1	YES (haemocytes)	1	> 60 days [41]	No or low dispersal [41]	1
A. mellifera	[12]	Bacteria	none	*ni*	-	YES (protein)[1]	1	> 60 days [41]	No or low dispersal [41]	1
Hemiptera										
M. persicae	[13]	Parasitoid	same as F0	NO (survival*)	0	*ni*	-	≤ 60 days [42]	No or low dispersal [59]	0
Diptera										
D. melanogaster	[14]	Bacteria	same as F0	NO (survival*)	0	*ni*	-	≤ 60 days [43]	Strong dispersal [60]	0
Ae. aegypti	[15]	Sephadex beads	same as F0	*ni*	-	NO (melanization)	0	≤ 60 days [44]	Strong dispersal [61]	0
A. gambia	[16]	Fungus	Apicomplexa	YES (prevalence)	1	*ni*	-	≤ 60 days [45]	Strong dispersal [62]	1
A. coluzzii	[17]	Apicomplexa	same as F0	NO (burden)	0	*ni*	-	≤ 60 days [45]	Strong dispersal [62]	0
Cx. pipiens	[18]	Apicomplexa	same as F0	NO (prevalence, burden)	0	*ni*	-	≤ 60 days [46]	Strong dispersal [63]	0
Coleoptera										
T. mollitor	[19]	LPS	same as F0	YES (survival)	1	YES (ab activity)	1	> 60 days [47]	No or low dispersal [22]	1
T. mollitor	[20]	LPS	same as F0	*ni*	-	YES (PO activity, haemocyte[2])	1	> 60 days [47]	No or low dispersal [22]	1
T. mollitor	[21]	LPS	none	*ni*	-	YES (ab activity)	1	> 60 days [47]	No or low dispersal [22]	1

T. mollitor	[22]	LPS	none	*ni*	-	YES (ab activity)	1	> 60 days [47]	No or low dispersal [22]	1
T. mollitor	[23]	K Bacteria or K Fungus or K Yeast	Bacteria	*ni*	-	YES (ab activity)	1	> 60 days [47]	No or low dispersal [22]	1
T. castaneum	[24]	HK Bacteria	Bacteria	YES (prevalence[2], survie[2])	1	YES (PO activity[2], ab activity[2])	1	> 60 days [48]	No or low dispersal [64]	1
T. castaneum	[25]	HK Bacteria	Bacteria	YES (survie[2])	1	YES (PO activity[2] , Immune transcripts[2])	1	> 60 days [48]	No or low dispersal [64]	1
T. castaneum	[26]	HK Bacteria	Bacteria	YES (survival)	1	*ni*	-	> 60 days [48]	No or low dispersal [64]	1
T. confusum	[26]	HK Bacteria	Bacteria	YES (survival)	1	*ni*	-	> 60 days [49]	No or low dispersal [65]	1
A. glabripennis	[27]	HK Bacteria or HK Fungus or Fungus	Fungus	YES (survival)	1	*ni*	-	> 60 days [50]	No or low dispersal [66]	1
Lepidoptera										
T. ni	[28]	Bacteria	none	*ni*	-	YES (PO activity , Immune transcripts)	1	> 60 days [51]	Strong dispersal [67]	1
T. ni	[29]	Virus	same as F0 / none	No (survival)	0	NO (haemocytes)	0	> 60 days [51]	Strong dispersal [67]	0
P. interpunctella	[30]	Virus	same as F0	YES (prevalence)	1	*ni*	-	> 60 days [52]	No or low dispersal [68]	1
P. interpunctella	[31]	Bacteria or Fungus	Bacteria and/ or Fungus	YES (survival[3])	1	*ni*	-	> 60 days [52]	No or low dispersal [68]	1
M. sexta	[32]	PGN	none / PGN	*ni*	-	YES (PO activity, ab activity)	1	≤ 60 days [53]	Strong dispersal [69]	1
M. sexta	[33]	PGN	Parasitoid	YES (prevalence)	1	YES (PO activity, ab activity)	1	≤ 60 days [53]	Strong dispersal [69]	1
G. mellonella	[34]	Bacteria	none	*ni*	-	YES (Immune transcripts)	1	> 60 days [54]	No or low dispersal [70]	1
Orthoptera										
T. oceanicus	[35]	Bacteria	none	*ni*	-	YES (Litic activity)	1	> 60 days [55]	ni	1

ab antibacterial, PO: phenoloxydase, HK: heat-killed, LPS: lipopolysaccaride (*E coli*),
PGN: peptidoglycan, ni: not investigated. [1] Exploration of biological mechanisms involved in TGIP. [2] Paternal immune priming. [3] Only for one combination: Mother and offspring exposed to fungus and offsrping reared on the good diet. * Parameter decrease.

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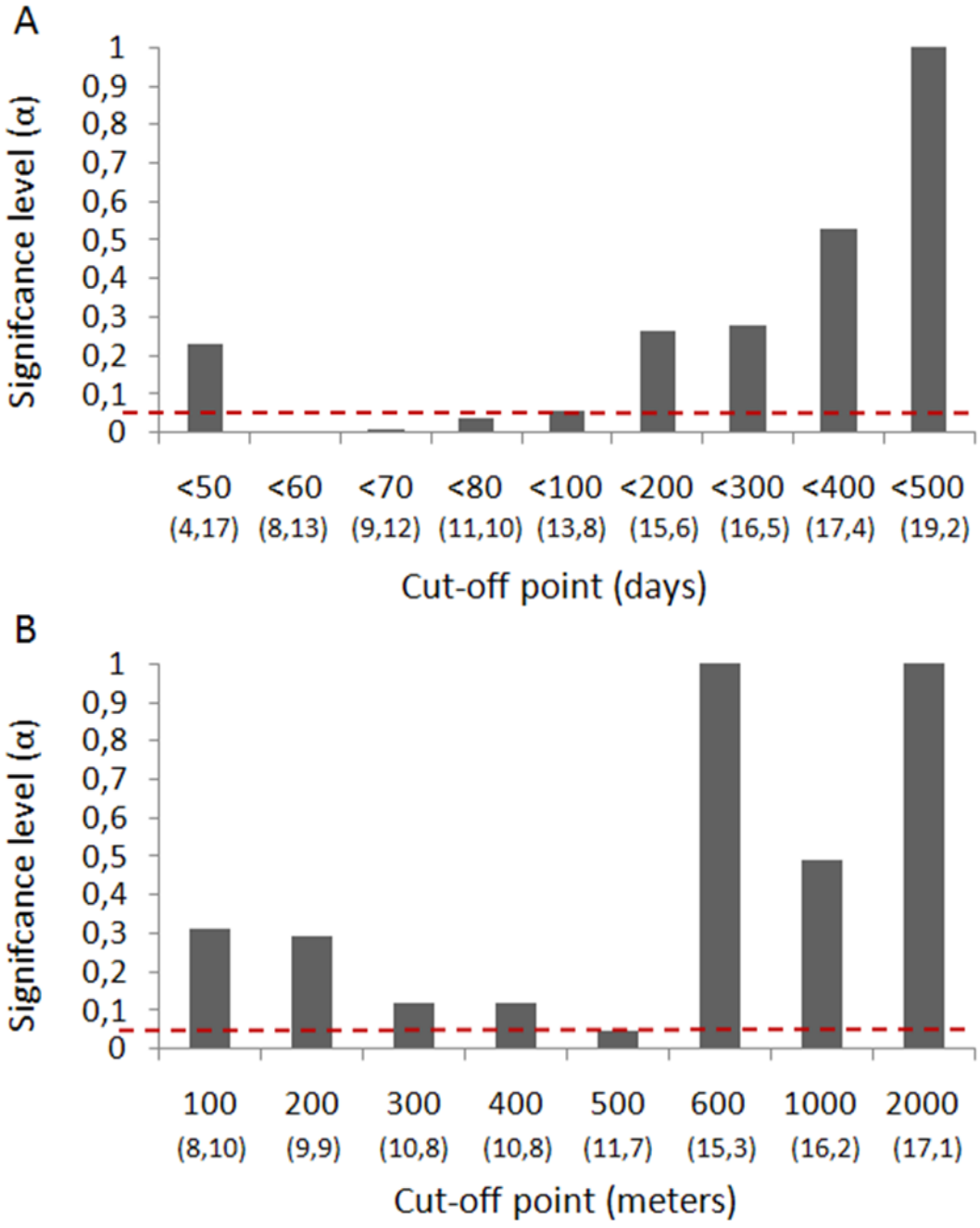

Figure S1: Control of the robustness of Overall Transgenerational Protection (OTP) analyses by using several different cut-off points: 9 points for longevity (A) and 8 for dispersal (B). Red dashed line represents the (α = 0.05) limit for Fisher Exact Test significance. The number of replicates either side of the cut-off point (inferior, superior) is written in parentheses.

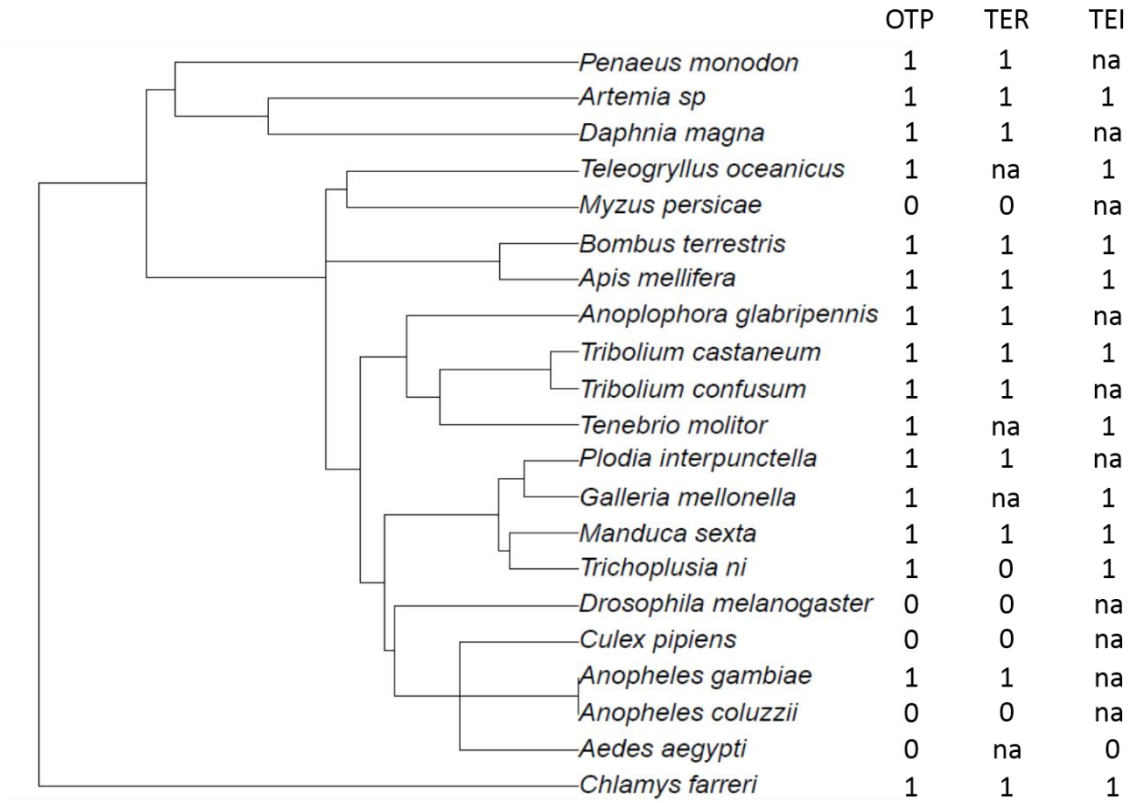

Figure S2: Phylogenetic associations between the 21 species of invertebrate included in the comparative analysis with their respective TER, TEI and OTP score.